DECLARATION OF JARED P. DEMPSEY, PHD INDEX OF EXHIBITS

10	Bell, Glenn B. et al., *Frequency-specific Responses in the Human Brian Caused by Electromagnetic Fields*, 123 JOURNAL OF THE NEUROLOGICAL SCIENCES 26-32 (1994)	000162-168
11	Cook, Charles M. et al., *Resting EEG Effects During Exposure to a Pulsed ELF Magnetic Field*, 26 BIOELECTROMAGNETICS 367-76 (2005)	000169-178
12	Arns, Martijn et al., *Electroencephalographic, Personality, & Executive Function Measures Associated with Frequent Mobile Phone Use*, 117 Int'l Journal of Neuroscience 1341-60 (2007; received June 1, 2006	000179-198
13	Klimesch, Wolfgang et al, SHORT COMMUNICATION: *Enhancing cognitive performance with repetitive transcranial magnetic stimulation at human individual alpha frequency*, 17 EUROPEAN JOURNAL OF NEUROSCIENCE, 1129-33 (2003)	000199-203
14	U.S. Pat. App. Pub. 2005/0256539A1 (Nov. 17, 2005)	000204-227
15	Yi Jin et al., *Therapeutic Effects of Individualized Alpha Frequency Transcranial Magnetic Stimulation (αTMS) on the Negative Symptoms of Schizophrenia,* 32 SCHIZOPHRENIA BULLETIN 556-61 at 556 (Oct. 27, 2005)	000228-233
16	Horvath, Jared C. et al, *Transcranial Magnetic Stimulation: A Historical evaluation and future prognosis of therapeutically relevant ethical concerns*, 37 JOURNAL OF MEDICAL ETHICS 137-43	000234-240
17	Lisanby, Sarah H., *Transcranial Magnetic Stimulation in Psychiatry: historical reflections and future directions*, 19 BIOLOGICAL PSYCHIATRY 486-90 (Mar. 15, 2024)	000241-243
18	Geller et al., *Slow Magnetic Stimulation Of Prefrontal Cortex In Depression and Schizophrenia*,	000244-249

	21 PROGRESS IN NEURO-PSYCHOPHARMACOLOGY & BIOLOGICAL PSYCHIATRY 105-10 (1997)	
19	McLain, Natalie J. et al., *Analytic Consistency and neural correlates of peak alpha frequency in the study of pain*,368 JOURNAL OF NEUROSCIENCE METHODS 3-4 (2021))	000250-263
20	Klimesch, Wolfgang, *EEG Alpha and Theta Oscillations Reflect Cognitive and Memory Performance: a review and analysis*, 29 Brain Research Reviews, 169-95 (1999)	000264-290
21	Notbohm, Annika et al., *Modification of Brain Oscillations via Rhythmic Light stimulation Provides Evidence for Entrainment but Not for Superposition of Event-Related Responses*, 10 FRONTIERS IN HUMAN NEUROSCIENCE Article 10 (2016)	000291-302
22	Ding, Nai et al., *Cortical entrainment to continuous speech: functional roles and interpretations*, 8 FRONTIERS IN HUMAN NEUROSCIENCE Article 311 (2014)	000303-309
23	Thaut, Michael H. et al, *The discovery of human auditory-motor entrainment and its role in the development of neurologic music therapy*, 217 PROGRESS IN BRAIN RESEARCH Chap. 13, 253-66 (2015)	000310-323
24	Okamura, Hisataka et al., *EEG Modification Induced by Repetitive Transcranial Magnetic Stimulation*, 18 JOURNAL OF CLINICAL NEUROPHYSIOLOGY 318-25 (2001)	000324-331
25	Thut, Gregor et al., *Rhythmic TMS causes local entrainment of natural oscillatory signatures*, 21 CURRENT BIOLOGY 1176-85 (2011)	000332-341
26	Faller, Josef et al., *Daily prefrontal closed-loop repetitive transcranial magnetic stimulation (rTMS) produces progressive EEG quasi-alpha phase entrainment in depressed adults*, BRAIN STIMUL. (2022)	000342-375

27	Tooley, Michael, *Electronic Circuits, Fundamentals and Applications*, at 74-75 (Newnes 2d. ed. 2002)	000376-382
28	Jin, Yi et al., *Alpha EEG Selectivity Predicts Efficacy Of RTMS In Schizophrenia*, ABSTRACTS OF THE XX INTERNATIONAL CONGRESS ON SCHIZOPHRENIA RESEARCH at 455 (2005)	000383-385
29	Jin, Y. et al., *Improvement in Alpha EEG Selectivity and Negative Symptoms in Schizophrenia Following rTMS Treatment*, 35:4 CLINICAL EEG & NEUROSCIENCE 224-25 (Wheaton 2004)	000386-387

EXHIBIT 1

Curriculum Vitae
Jared P. Dempsey, Ph.D.

Cellular: (405) 269-3440
E-Mail: dempsey@recoveryscience.org
Address: 6405 107th, Ste 100
Lubbock, TX 79424

CURRENT APPOINTMENT

Chief Scientist
Trac9.com, LLC
Lubbock, TX

EDUCATIONAL BACKGROUND

08/07 –08/08 **NIAAA Postdoctoral Research Fellow**
Center for Drug and Alcohol Programs
Medical University of South Carolina
Research Preceptors: Carrie L. Randall, Ph.D.
Suzanne E. Thomas, Ph.D.

08/07 **Doctorate of Philosophy in Clinical Psychology**
Texas Tech University, Lubbock, Texas
Dissertation Title: Smoking-Cue Modulation of the Startle Reflex and the Relationship to Stages of Change.
Research Preceptor: Lee M. Cohen, Ph.D.

07/06 –08/07 **Pre-Doctoral Internship in Clinical Psychology**
Charleston Consortium Psychology Internship Program
Medical University of South Carolina
Research Preceptors: Carrie L. Randall, Ph.D.
Suzanne E. Thomas, Ph.D.

06/04 **Masters of Arts, Psychology**
Texas Tech University, Lubbock, Texas
Thesis Title: Predicting School Maladjustment in Peer-Victimized Children
Research Preceptor: Gary D. Fireman, Ph.D.

05/02 **Bachelor of Arts, Psychology**
California State University, Long Beach, California
Honors: Cum Laude

05/00 **Associate in Arts**
Orange Coast College, Costa Mesa, California
Honors: Phi Alpha Mu

PROFESSIONAL APPOINTMENTS

2016 – Present Chief Scientist & Co-Founder
Trac9.com
Lubbock, TX

2024 – Present Chief Science Officer & Co-Founder
Recovery Science, LLC
Lubbock, TX

2014 – 2024 Chief Science Officer & Co-Founder
NLW Partners, LLC
Lubbock, TX

2013 – 2014 Assistant Professor
Addictive Disorders and Recovery Studies Program
Department of Community, Family, and Addiction Services
Texas Tech University, Lubbock, TX

2011 – 2014 Core Faculty Member
Doctoral Program in Clinical Psychology
Fielding Graduate University, Santa Barbara, CA

2010 – 2011 Director, Psychological Services & Research
Center for Tobacco Research and Prevention (C-TRP)
Seretean Wellness Center
Oklahoma State University, Stillwater, OK

2008 – 2011 Assistant Professor
Department of Psychology
Oklahoma State University, Stillwater, OK

2007 – 2008 Post-Doctoral Fellow
Center for Drug and Alcohol Problems
Medical University of South Carolina, Charleston, SC

2007 – 2008 Adjunct Faculty
Department of Psychology
The Citadel, Charleston, SC

2006 – 2008	Adjunct Faculty *Department of Psychology* College of Charleston, Charleston, SC
2006 – 2007	Clinical Psychology Intern *National Crime Victims Center* Medical University of South Carolina, Charleston, SC
2003 – 2006	Graduate Research Assistant *Graduate Psychology Education Grant (HRSA)* Texas Tech University, Lubbock, TX
2003 – 2006	Graduate Teaching Assistant *Department of Psychology* Texas Tech University, Lubbock, TX
2004 – 2006	Graduate Research Assistant & Clinical Intern *Neuropsychiatry Department (Dr. Patricia Sutker)* Texas Tech University Health Sciences Center, Lubbock, TX
2002 – 2004	Graduate Research Assistant *Behavioral Pharmacology Laboratory (Dr. Lee Cohen)* Texas Tech University, Lubbock, TX
2002 – 2004	Research Assistant *Violence Prevention Laboratory (Dr. Gary Fireman)* Texas Tech University, Lubbock, TX
2000 – 2002	Applied Behavioral Analysis Therapist *Lovaas Institute for Early Intervention* Las Angeles, CA
2000 – 2002	Information Technology Specialist *Virtual Microscopy Neuroscience Laboratory* California State University, Long Beach, CA

SERVICE ACTIVITIES

2012 – 2014	Chair, Educational Technology Committee School of Psychology Fielding Graduate University, Santa Barbara, CA
2005 – 2014	Editorial Board: *Journal of Psychopathology and Behavioral Assessment*
2009 – 2011	Board Member: *Tobacco & Health Advisory Board* Oklahoma State University, Stillwater, OK

2009 – 2011 Faculty Advisor: *Psi Chi - The International Honor Society in Psychology*
Oklahoma State University, Stillwater, OK

2008 – 2010 Committee Member: The Committee to Promote Student Interest
The Society for Psychophysiological Research

2008 – 2010 Committee Member: Turskey Travel Award
The Society for Psychophysiological Research

AWARDS/HONORS

- 2014: Honored Faculty Mentor, Phi Beta Kappa
  Texas Tech University, Lubbock, TX
- 2009: Supportive Faculty Award, Non-Traditional Student Organization
  Oklahoma State University, Stillwater, OK
- 2009: College of Arts and Sciences Summer Research Funding and Travel
  Oklahoma State University, Stillwater, OK
- 2009: College of Arts and Sciences Travel Award
  Oklahoma State University, Stillwater, OK
- 2008: Minority Researcher Grantsmanship Travel Award
  National Institute of Neurological Disorders and Stroke
  National Institutes of Health, Bethesda, MD
- 2007: Best Psychology Intern Research Paper
  Institute of Psychiatry and Behavioral Sciences
  Medical University of South Carolina, Charleston, SC
- 2006: Exceptional Graduate Research in Clinical Psychology
  Texas Tech University, Lubbock, TX
- 2005: Clay E. George Endowed Scholarship
  Texas Tech University, Lubbock, TX
- 02-06: Department of Psychology Competitive Scholarship (2002, 04, 05, 06)
  Texas Tech University, Lubbock, TX

SECURED GRANTS & FUNDRAISING

Functional Near-Infrared Assessment of Addiction Recovery
Funding Agency: **The National Foundation for Collegiate Recovery**
Southlake, TX
Principal Investigator: **Jared P. Dempsey**
Funding Dates: 2014 – 2015
Total Direct Costs: $94,161

Development of a Biological Marker of Addiction
Funding Agency: **Office of Research**
College of Human Sciences
Texas Tech University, Lubbock, TX

Principal Investigator: **Jared P. Dempsey**
Funding Dates: 2013 – 2014
Total Direct Costs: $5,000

Research & Publications Associated with Smoke-Free Campus Status
Funding Agency: **Sub Award - Tobacco Settlement Endowment Trust**
Seretean Wellness Center, Oklahoma State University
Oklahoma State University, Stillwater, Oklahoma
Principal Investigator: **Jared P. Dempsey**
Funding Dates: July 2010 – June 2011
Total Direct Costs: $15,686

Efficacy and Perception Change Associated with Smoke-Free Campus Status
Funding Agency: **Sub Award - Tobacco Settlement Endowment Trust**
Seretean Wellness Center, Oklahoma State University
Oklahoma State University, Stillwater, Oklahoma
Principal Investigator: **Jared P. Dempsey**
Funding Dates: September 2009 – June 2010
Total Direct Costs: $16,796

Science-Based Smoking Cessation Kits for Oklahoma State University
Funding Agency: **GlaxoSmithKline, PLC**
Philadelphia, PA
Principal Investigator: **Jared P. Dempsey**
Funding Date: October 2009
Total Direct Costs: 500 GSK Quit Kits

Kick-Tobacco: A Self-help Tobacco Cessation Kit
Funding Agency: **Seretean Wellness Center, Oklahoma State University**
Oklahoma State University, Stillwater, Oklahoma
Principal Investigator: **Jared P. Dempsey**
Funding Dates: September 2009 – June 2010
Total Direct Costs: $6,650

International Research Presentation Funding: Student Exposure to Science
Location: The Society for Psychophysiological Research, Berlin, Germany
Funding Agency: **Oklahoma State University Foundation**
Stillwater, Oklahoma
Principal Investigator: **Jared P. Dempsey**
Funding Date: October 2009
Total Direct Costs: $5,000

International Research Presentation Funding: Student Exposure to Science
Location: The Society for Psychophysiological Research, Berlin, Germany
Funding Agency: **Office of the Vice President for Research and Technology Transfer**
Oklahoma State University, Stillwater, Oklahoma

Principal Investigator: **Jared P. Dempsey**
Funding Date: October 2009
Total Direct Costs: $5,000

SUPERVISED GRANTS & AWARDS

CALUE Undergraduate Research Award
Center for Active Learning & Undergraduate Engagement, Texas Tech University
Study: Depression and PFC Activity in Addiction Recovery
Awarded Scholar: Ryan A. Burden
Supervising Scientist: **Jared P. Dempsey, Ph.D.**
Funding Dates: September 2014
Total Direct Costs: $1,080

CALUE Undergraduate Research Award
Center for Active Learning & Undergraduate Engagement, Texas Tech University
Study: Frontal Lobe Functioning in Successful Addiction Recovery
Awarded Scholar: C. Jarod Herrera
Supervising Scientist: **Jared P. Dempsey, Ph.D.**
Funding Dates: January 2014
Total Direct Costs: $1,080

Morris K. Udall & Stewart L. Udall Foundation Scholarship
Study: Culturally Sensitive Smoking Cessation Treatment for Native Americans
Awarded Scholar: Brooke N. Hill
Supervising Scientist: **Jared P. Dempsey, Ph.D.**
Funding Dates: August 2010
Total Direct Costs: $5,000

Lew Wentz Research Scholar Award
Study: Neurologically Assessed Smoking Cessation and the Role of Psychopathic Traits
Office of Scholar Development and Recognition, Oklahoma State University
Awarded Scholar: Stephanie Kline
Supervising Scientist: **Jared P. Dempsey, Ph.D.**
unding Dates: August 2010 – July 2010
Total Direct Costs: $4,500

Niblack Research Scholar
Study: Image Orientation and Physiologically-Assessed Appetitive Nature of Drug Cues
Office of the Vice President for Research and Technology, Oklahoma State University
Awarded Scholar: Brooke N. Hill
Supervising Scientist: **Jared P. Dempsey, Ph.D.**
Funding Dates: August 2009 – July 2010
Total Direct Costs: $10,100

Lew Wentz Research Scholar Award
Study: Depression Severity as a Moderating Variable in Drug Cue Appetitive Responding
Office of Scholar Development and Recognition, Oklahoma State University
Awarded Scholar: David E. Lovett
Supervising Scientist: **Jared P. Dempsey, Ph.D.**
Funding Dates: August 2009 – July 2010
Total Direct Costs: $4,500

Lew Wentz Research Scholar Award
Study: Electromyography Assessed Gastric Impairment in Recovered Eating Disorders
Office of Scholar Development and Recognition, Oklahoma State University
Awarded Scholar: Devon E. Eldridge
Supervising Scientist: **Jared P. Dempsey, Ph.D.**
Funding Dates: August 2009 – July 2010
Total Direct Costs: $4,500

GRANT SUBMISSIONS & ACTIVITY

Contingency Management and Prefrontal Cortex Activation Changes in Alcohol Use Disorder: Delay Discounting as a Mechanism of Change.
Funding Agency: **National Institute on Alcohol Abuse and Alcoholism (PA-15-301)**
Role: **Co-PI**
Proposed Funding Dates: 2015-2017
Total Project Funds Requested: $275,000
Status: Under Review

A Bio-Behavioral Model of Financial Misbehavior Drawing from Behavioral Theories of Incentive Salience and Personality Theories of Empathy.
Funding Agency: **National Science Foundation (Proposal 14-0027)**
Interdisciplinary Behavioral and Social Science Research (IBSS)
Role: **Subcontracting PI**
Proposed Funding Dates: 2014-2016
Requested Subcontract Funds: $45,000
Total Project Funds Requested: $203,850
Status: Unfunded

National Science Foundation (Proposal 7216806)
Functional Near-Infrared Spectroscopy Assessment of Reading Acquisition in Hispanic Children
Role: **Co-PI**
Proposed Funding Dates: 2012-2013
Requested Funds: $500,000
Status: Rejected for review due to administrative error during FastLane submission

Sarkeys Foundation
Study: Mobile Mental Health for Rural Populations
Role: **PI/Applicant**
Proposed Funding Dates: 2009-2011
Requested Funds: $371,305
Status: Selected for Funding. All projects funds diverted for funding round.

The Foundation for Alcohol Research (ABMRF)
Study: Psychological Response to Drug Cues as Treatment Effect in Alcohol Dependence
Role: **PI/Applicant**
Proposed Funding Dates: 2009-2011
Requested Funds: $250,000
Status: Scored, Unfunded

Oklahoma Center for the Advancement of Science & Technology (OCAST)
Study: A New Test of Smoking Cessation Treatment Effectiveness
Role: **PI/Applicant**
Proposed Funding Dates: 2009-2012
Requested Funds: $230,000
Status: Unfunded

National Institute on Alcohol Abuse & Alcoholism (NIH NIAAA 1 R21AA018165-01)
Psychological Response to Drug Cues as Treatment Effect in Alcohol Dependence
Role: **PI/Applicant**
Proposed Funding Dates: 2009-2011
Requested Funds: $250,000
Status: Scored, Unfunded

PROFESSIONAL AFFILIATIONS

- American Psychological Association
- Division of Psychopharmacology and Substance Abuse: American Psychological Association
- Research Society on Alcoholism
- Sigma Xi – The Scientific Research Society
- Society for Psychophysiological Research

PUBLICATIONS (Peer-Refereed)

Rodriguez-Menendez, G., **Dempsey, J.P.**, Albizu, T., Power, S., & Wilkerson, M.C. (2017). Faculty and Student Perceptions of Clinical Training Experiences in Professional Psychology. *Training and Education in Professional Psychology*, 11(1): 1-9.

Dempsey, J.P., Harris, K.S., Shumway, S.T., Kimball, T.G., Herrera, J.C., Bradshaw, S., Dsauza, C.M. (2015). Functional Near Infrared Spectroscopy as a Potential Biological Assessment of Addiction Recovery: Preliminary Findings. *The American Journal of Drug and Alcohol Abuse,* 41(2):119-26.

Lechner, W., Grant, D., Meier, E., Mills, A., Judah, M., & **Dempsey, J.P.** (2014). The Influence of Stress on the Affective Modulation of the Startle Response to Nicotine Cues. *Applied Psychophysiology and Biofeedback, 39*(3-4), 279-285. doi: 10.1007/s10484-014-9266-5

Dempsey, J. P., Cohen, L. M., Watson, N. L., Lechner, W. V., Hobson, V. L., & Smith, K. (2011). The Association of Blood Pressure and the Risk of Alcohol Use Disorders Among Smokers: Implications for Screening and Treatment. *Alcoholism Treatment Quarterly, 29*(2), 123-131.

Dempsey, J. P., & Cohen, L. M. (2010). Commentary on Hajek et al. (2010): Investigating the stress reduction in smoking cessation. *Addiction, 105*(8), 1472-1473.

Book, S. W., Thomas, S. E., **Dempsey, J. P.**, Randall, P. K., & Randall, C. L. (2009). Social anxiety impacts willingness to participate in addiction treatment. *Addictive Behaviors*, 34(5), 474-476.

Dempsey, J. P., Randall, P. K., Thomas, S. E., Book, S. W., & Carrigan, M. H. (2009). Treatment of social anxiety with paroxetine: mediation of changes in anxiety and depression symptoms. *Comprehensive Psychiatry, 50*(2), 135-141.

Dempsey, J. P., Back, S. E., Waldrop, A. E., Jenkins, L., & Brady, K. T. (2008). The Influence of Monetary Compensation on Relapse among Addicted Participants: Empirical vs. Anecdotal Evidence. *American Journal on Addictions, 17*(6), 488 - 490.

Morrell, H. E. R., Cohen, L. M., & **Dempsey, J. P.** (2008). Smoking prevalence and awareness among undergraduate and health care students. *The American Journal on Addictions*, 17(3), 181-186.

Dempsey, J. P., Cohen, L. M., Hobson, V. L., & Randall, P. K. (2007). Appetitive nature of drug cues re-confirmed with physiological measures and the potential role of stage of change. *Psychopharmacology, 194*(2), 253-260.

Dempsey, J. P., Fireman, G. D., & Wang, E. M. (2006). Transitioning out of peer victimization in school children: gender and behavioral characteristics. *Journal of Psychopathology and Behavioral Assessment*, 28(4), 271-280.

PUBLICATIONS (Non-Refereed)

Davis, M., Duncan, N., & **Dempsey, J. P.** (2011) International Drug Policies: Sanctions and Economic Assistance. In M. Kleiman & J. Howard (Eds.), *Encyclopedia of Drug Policy: The War on Drugs Past and Present*. Thousand Oaks, Calif.: Sage Publications.

Ardrey, T., Hill, B.N., Kline, S. & **Dempsey, J. P.** (2011) Oklahoma Drug Law & Policy. In M. Kleiman & J. Howard (Eds.), *Encyclopedia of Drug Policy: The War on Drugs Past and Present*. Thousand Oaks, Calif.: Sage Publications.

Ardrey, T., Hill, B.N., Kline, S. & **Dempsey, J. P.** (2011). Presidential Commission on Narcotics and Drug Abuse. In M. Kleiman & J. Howard (Eds.), *Encyclopedia of Drug Policy: The War on Drugs Past and Present*. Thousand Oaks, Calif.: Sage Publications.

Dempsey, J. P. (2009, December 01). Consultation in oral surgery & medicine: Quitting chewing tobacco - The Great American Spit-out. *Dear Doctor*, 3, 54-55.

Amstadter, A. B., **Dempsey, J. P.**, Watson, N. L., & Hubel, G. S. (2009). Co-occurring anxiety disorders and substance use disorders. In G. L. Fisher & N. A. Roget (Eds.), *Encyclopedia of substance abuse prevention, treatment, and recovery*. Thousand Oaks, Calif.: Sage Publications.

Dempsey, J. P., Amstadter, A. B., Hubel, G. S., & Watson, N. L. (2009). Cognitive behavioral therapy in substance use disorders. In G. L. Fisher & N. A. Roget (Eds.), *Encyclopedia of substance abuse prevention, treatment, and recovery*. Thousand Oaks, Calif.: Sage Publications.

Hobson, V. L., & **Dempsey, J. P.** (2009). Alcohol related birth defects. In G. L. Fisher & N. A. Roget (Eds.), *Encyclopedia of substance abuse prevention, treatment, and recovery*. Thousand Oaks, Calif.: Sage Publications.

Dempsey, J. P., Fireman, G. D., & Fireman, S. E. (2005). Elder neglect. In S. Gelman, L. Marigolds, A. Huston, M. Zimmerman, M. E. Lachman, W. Dunn, T. Saxon, J. Raths, R. Thompson & M. Thompson (Eds.), *The Encyclopedia of Human Development*. Thousand Oaks, CA: SAGE Publications.

PROFESSIONAL PRESENTATIONS

Dempsey, J. P. (2019, October). The Importance of Standardized Assessments: Predicting Treatment Failure and Success. Keynote Address at the Oklahoma Drug & Alcohol Professional Counselor Association Annual Conference, Norman, OK.

McCabe, P., **Dempsey, J. P.**, King, B., & Yingst (2019, August), J. Vaping and Smoking Cessation – Can You Use an E-Cig to Quit Smoking? Do Treatment Programs offer them as an alternative or should they be banned? Symposium conducted at the National Conference on Addiction Disorders – East, Baltimore, MD.

Dempsey, J. P. & Rauber, R. (2019, April). The Importance of Collegiate and Young Adult Substance Use Disorder Treatment in the Justice System. Symposium at the Texas Association of Specialty Courts Annual Conference, Galveston, TX.

Dempsey, J. P. (2019, April). The Importance of Standardized Assessments & Outcomes for Successful Addiction Recovery. Keynote Address at the Annual Conference of Texas Association of Addiction Professionals, Fort Worth, TX.

Harris, K. S. & **Dempsey, J. P.** (2016, April). Resilience, Recovery, and Neuroscience: Why Collegiate Recovery Works. Symposium conducted at the 7th Annual National Collegiate Recovery Conference, Atlanta, GA.

Rodriguez-Menendez, G., **Dempsey, J. P.**, Albizu, T., Bridges-Power, S., & Haldeman, D. (2015, August). Innovations in the Science and Art of Clinical Supervision. Symposium conducted at the Annual Convention of the American Psychological Association, Toronto, ON, Canada.

Laban, M. & **Dempsey, J. P.** (2014, August). Assessment of ADHD in Clinical Practice. Poster presented at the Annual Convention of the American Psychological Association, Washington, D.C.

Dempsey, J. P. (2014, April). The Neuroscience of Addiction and Recovery. Invited guest presentation at Kennesaw State University's Center for Young Adult Addiction and Recovery's Pathway to Understanding Conference, Kennesaw, GA.

Dempsey, J. P. (2014, February). Addiction Recovery and Advances in Biological Markers. Invited guest presentation at Texas Tech University's Addiction Recovery Conference, Lubbock, TX.

VanderVeen, J., Watson, N. L., Sawyer, H., Hanft, A., **Dempsey, J. P.**, & Cohen, L. (2010, November). The influence of binge drinking and impulsivity on cue response among nicotine dependent college students. Poster presented at the 44th Annual Convention of the Association for Behavioral and Cognitive Therapies, San Francisco, CA.

Eldridge, E. D., **Dempsey, J. P.**, Chee, L., Kline, S. & Cavett, C. (2010, October). The Effects of Eating Disorders on Gastric Motility. Poster accepted for presentation at the annual meeting of the Society for Psychophysiological Research, Portland, Oregon.

Dempsey, J. P. (2010, July 16th). The college smoker, mental health, and current advances in tobacco research techniques. Invited Presentation for the Oklahoma State Regents for Higher Education and Community Partners – Tobacco Free College Symposium, Midwest City, OK.

Claborn, K. R., **Dempsey, J. P.**, Jaquez, S. D., Junghans, A. N., Hill, B. N., Lechner, W. V., Cohen, L.M. & al'Absi, Mustafsa (2009, October). Cotinine as a potential predictor of increased

anxiety during nicotine withdrawal. Poster presented at the annual meeting of the Society for Psychophysiological Research, Berlin, Germany.

Eldridge, E. D., **Dempsey, J. P.**, Hughes, S.T., Lovett, D. E., Hornyik, C. D. & Cohen, L.M. (2009, October). Social deviance and startle modulation: Different results for different measures of psychopathy. Poster presented at the annual meeting of the Society for Psychophysiological Research, Berlin, Germany.

Hornyik, C. D., **Dempsey, J. P.**, Lovett, D. E., Eldridge, E. D., Junghans, A. N., Grigsby, R. G., Hill, B. N., Abramson C. I. & Cohen, L. M. (2009, October). Affective range vs. mean: A novel method for understanding atypical affective modulation of the startle reflex. Poster presented at the annual meeting of the Society for Psychophysiological Research, Berlin, Germany.

Jaquez, S. D., **Dempsey, J. P.**, Junghans, A. N., Claborn, K. R., Kaplan, J. M., Lechner, W. V. & Cohen, L.M. (2009, October). Cardiovascular activity and process of change in nicotine withdrawal. Poster presented at the annual meeting of the Society for Psychophysiological Research, Berlin, Germany.

Junghans, A. N., **Dempsey, J. P.**, Claborn, K. R., Jaquez, S.D., Lovett, D. E., Grigsby, R. G., Lechner, W. V., & Cohen, L.M. (2009, October). Independent contribution of anxiety in nicotine withdrawal-induced cardiac deceleration. Poster presented at the annual meeting of the Society for Psychophysiological Research, Berlin, Germany.

Lovett, D. E., **Dempsey J. P.**, Hornyik, C. D., Junghans, A. N., Eldridge, E. D., Kaplan, J. M. & Cohen, L. M. (2009, October). Atypical affective modulation of startle and symptoms of depression. Poster presented at the annual meeting of the Society for Psychophysiological Research, Berlin, Germany.

Junghans, A. E., Lovett, D. E., Eldridge, E. D., Grant, D. M. & **Dempsey, J. P.** (2009, November). Smoking to cope with social anxiety: A preliminary investigation. Poster presented at the annual convention of the Association for Behavioral and Cognitive Therapies, New York, NY.

Dempsey, J. P., (2009, June). Federal grant navigation and funding opportunities for students. Invited workshop presented to the American Indians Into Psychology program, Oklahoma State University, Stillwater, OK.

Hobson, V. L., **Dempsey, J. P.**, Cohen, L. M. & Watson, N.L. (2008) Blood Pressure is Associated with Risk of Alcohol use Disorder in Smoking College Students. Poster presented at the annual meeting of the Society for Psychophysiological Research, Austin, Texas.

Dempsey, J. P., Thomas, S. E. & Randall, C. L. (2007) Drinking to cope before, during, and after social events: Gender differences in socially anxious alcoholics. *Alcoholism-Clinical and Experimental Research*, 31(6), 41A.

Bacchi, D. R., McDougal, K. M. & **Dempsey, J. P.** (2006, November) Utilization of quit kits among tobacco-using college students. Poster presented at the annual convention of the American Public Health Association, Boston, Massachusetts.

Dempsey, J. P., Hobson, V. L., Cohen, L. M. & Bacchi D. R. (2006, October) Appetitive nature of smoking-cues reconfirmed with physiological measures: The role of stage of change. Poster presented at the annual meeting of the Society for Psychophysiological Research, Vancouver, BC, Canada.

Hobson, V. L., **Dempsey, J. P.,** Cohen, L. M., Bacchi, D. R. & Jaeger, M. L. (2006, October) Order influences by picture type in affective modulation of the startle reflex: Does randomization remove all influence? Poster presented at the annual meeting of the Society for Psychophysiological Research, Vancouver, BC, Canada.

Humphreys, J., **Dempsey, J. P.**, Sutker, P. B. & O'Bryant, S. E. (2006, September) Convergent validity of the repeatable battery for the assessment of neuropsychological status in a memory disorder clinic sample. *Archives of Clinical Neuropsychology*, 21(6), 557-558.

Silva, M. E., Humphreys, J., **Dempsey, J. P.**, Sutker, P. B. & O'Bryant, S. E. (2006, September) Test-retest reliability of the repeatable battery for the assessment of neuropsychological status in a memory disorder clinic sample. *Archives of Clinical Neuropsychology*, 21(6), 560-561.

Dempsey, J. P., Hobson, V. L., Rhodes, A. A., Harris, J. E., Kerr, L. A. & Cohen, L. M. (2005, November) Differences in cortisol production at varying levels of nicotine deprivation. Poster presented at the annual convention of the Texas Psychological Association, Houston, Texas.

Dempsey, J. P., Davis, K. E., Hobson, V. L., Cohen, L. M. & Borrego, J. R. (2005, November). Changes in perception of underserved populations through a behavioral medicine practicum experience: The graduate psychology education program. Poster presented at the annual convention of the Association for Behavioral and Cognitive Therapies, Washington, DC.

PROFESSIONAL ACTIVITIES

09/14	Invited Reviewer: *Motivation & Emotion*
12/13	Invited Reviewer: *Nicotine & Tobacco Research*
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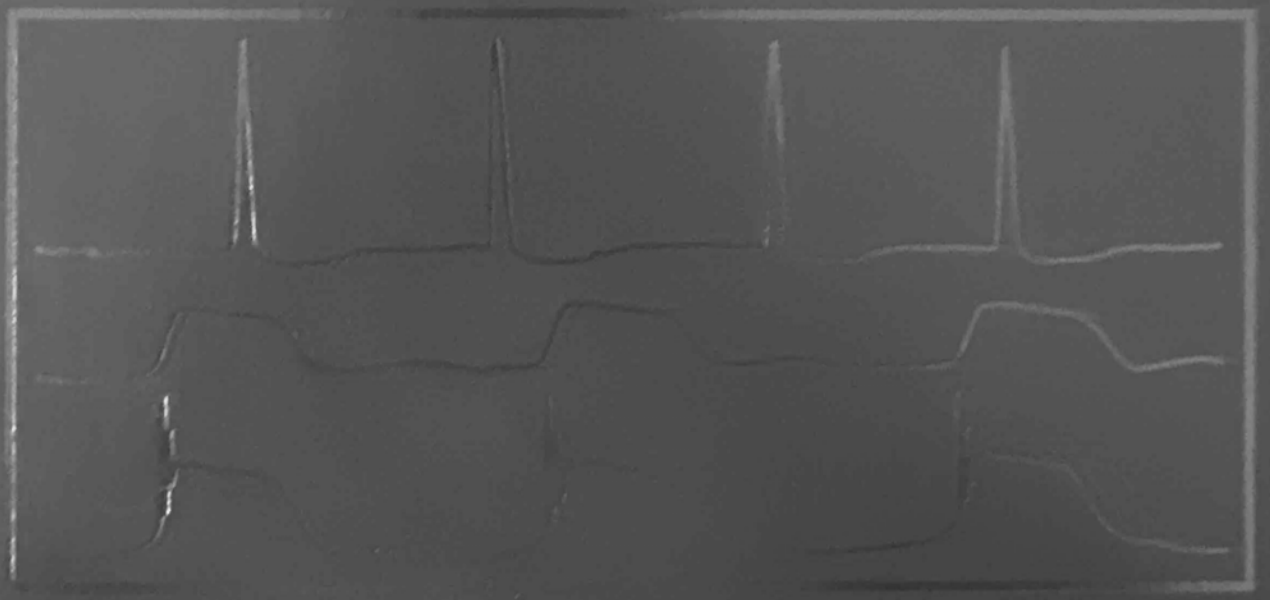
EXHIBIT 2

Electroencephalography

Basic Principles, Clinical Applications, and Related Fields

ERNST NIEDERMEYER / FERNANDO LOPES DA SILVA

FOURTH EDITION



Electroencephalography

Basic Principles, Clinical Applications, and Related Fields

FOURTH EDITION

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2. Introduction to the Neurophysiological Basis of the EEG and DC Potentials[1]

Erwin-Josef Speckmann and Christian E. Elger

The clinical electroencephalographer correlates central nervous system functions as well as dysfunctions and diseases with certain patterns of the electroencephalogram (EEG) on an empirical basis. Obviously, this method has been found valuable in clinical practice. Therefore, why should the clinical electroencephalographer study the basic elementary processes underlying the EEG? There is little doubt that the range of EEG interpretations can be much widened and misinterpretations avoided when the underlying elementary processes are also considered. This is true especially for convulsive disorders and cerebral metabolic disturbances. For example, an isoelectric EEG can be caused by selective pCO_2 increase while the brain is sufficiently supplied with O_2. On the other hand, in the presence of practically normal pCO_2 levels, cerebral hypoxia may be the cause. It will be pointed out below that the prognosis may be quite different in these two cases.

Elementary Processes of Extracellular Field Potential Generation

The basic mechanisms that give rise to potentials recorded outside the central nervous system (CNS) elements will be described. Such extracellular potentials are generally known as field "potentials" (Speckmann and Caspers, 1979a).

In the course of this presentation, the morphology of generator structures is discussed briefly. Then, the electrical activity demonstrable with intracellular recordings from neurons and glia cells is described. On the basis of this information, the principles of the generation of extracellular field potentials are outlined and the various types of field potentials are characterized.

Generator Structures

The central nervous system essentially consists of nerve cells and glia cells. The arrangement of neurons usually shows a specific type of laminar character. Glia cells are located between neurons.

As shown in Figure 2.1, several processes emerge from the nucleus-containing cellular soma (body) of the nerve cell. These processes can be divided into two types according to their function. Most of the processes are dendrites that branch off into numerous small ramifications. Every cell also has an axon that may split up into multiple collaterals. Such an axon provides contact with other nerve cells or with other target organs. In the case of interneuronal connections, the contact consists of synapses that cover the dendrites, the soma, and the axon hillock in large numbers. Thus, nerve cells are usually covered with several thousand synapses (Palay and ChanPalay, 1977).

The glia cells are imbedded between nerve cell somata, dendrites, and axons. They usually have several processes that make contact with somata and processes of nerve cells; they may also make contact with vessels. This histological arrangement results in a cerebral extracellular space consisting of very narrow intercellular clefts (De Robertis and Carrea, 1965).

Neuronal Activity Recorded Intracellularly

Next, those essential potentials that can be demonstrated with intracellular recordings are characterized briefly. When the membrane of the nerve cell body is penetrated by a microelectrode, a potential of about 60–70 mV with negative polarity in the intracellular space can be recorded. This membrane potential is subject to various fluctuations that are elicited chiefly by synaptic activities. Their mechanisms are shown in greater detail in Figure 2.2. As can be derived from this schematic illustration, the neuron from which the soma membrane potential is recorded has synaptic connections. The corresponding presynaptic structures are also explored with microelectrodes. If an action potential travels along the fiber, which ends in an excitatory synapse, an excitatory postsynaptic potential (EPSP) occurs in the following neuron (Fig. 2.2*A*). If two action potentials travel along the same fiber with a short interval, there will be a summation of EPSP triggering an action potential on the postsynaptic neuron after reaching the membrane threshold. If an action potential travels along a fiber ending in an inhibitory synapse, then hyperpolarization will occur, representing an inhibitory postsynaptic potential (IPSP) (Eccles, 1964; Hubbard et al., 1969; Shepherd, 1974).

Because of the time course of the various membrane potential fluctuations, the postsynaptic potentials are thought to contribute primarily to the generation of the extracellular field potentials in question (Creutzfeldt and Houchin, 1974; Hubbard et al., 1969; Speckmann and Caspers, 1979a; Speckmann et al., 1984). For this reason, the ional mechanisms of these potentials are discussed in greater detail. The individual events of this process are presented with a magnified time base (see Figure 2.4). With the elicitation of an EPSP, a net inflow of cations occurs across the subsynaptic membrane. This gives rise to depolarization of the subsynaptic membrane. As shown in Figure 2.2*B*, a potential gradie

[1] This chapter was translated from German by E. Niedermeyer.

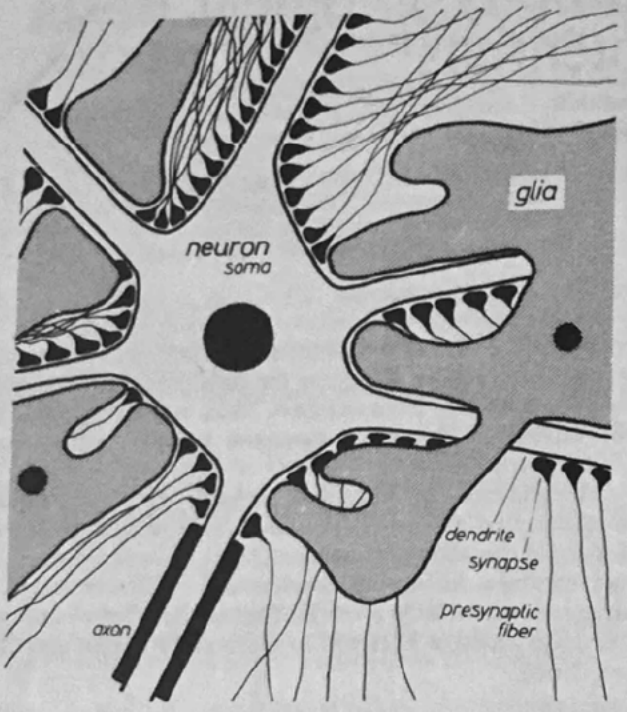

Figure 2.1. Schematic drawing of morphology and histology of neuronal and glial elements.

develops along the neuronal membrane in the intra- and extracellular space. Because of this potential gradient, cations move along the nerve cell membrane through the extracellular space in the direction of the subsynaptic region. An inversely directed flow takes place in the intracellular space. With the generation of an IPSP, there is an outflow of cations from the nerve cell and/or an inflow of anions into the nerve cell. These changes first increase the membrane potential at the subsynaptic membrane in comparison with the surrounding segments of the membrane. For this reason, a potential gradient develops along the nerve cell membrane, as in the case of the EPSP genesis. This potential gradient causes, in the extracellular space, a flow of cations from the subsynaptic region to the surrounding portions of the membrane. An inverse process develops in the intracellular space (Hubbard et al., 1969).

The ion fluxes in the extracellular space are of paramount significance in the generation of field potentials. Therefore, these processes are further discussed in the following chapters.

Glia Activity Recorded Intracellularly

In addition to the neurons, glial cells may also play a role in the generation of extracellular field potentials (Kuffler and Nicholls, 1966; Somjen and Trachtenberg, 1979). Therefore, the bioelectric properties of glial cells are summarized.

If a glia cell is penetrated with a microelectrode, a membrane potential can be recorded with a polarity similar to that of the nerve cells. The size of this membrane potential approximates the potassium equilibrium potential and hence somewhat exceeds the membrane potential of nerve cells. In contrast to neurons, glial cells fail to show any action potentials, and there are also no postsynaptic potentials. Thus, in contrast to neurons, glial cells do not show characteristic potentials that distinguish them unmistakably from other cells. The glial membrane potential, however, is also not constant. An augmentation of the extracellular potassium concentration (potassium activity) causes depolarization of glial cells (Fig. 2.3*A*). Concentration changes of other ions cause only negligible alterations of the glial cell membrane potential. The glial cell is hence comparable to a potassium electrode (Kuffler and Nicholls, 1966; Kuffler et al., 1966).

The dependency of the glial membrane potential on the extracellular potassium concentration is the reason for a functional linkage with adjacent neuronal structures. Neuronal activity is associated with outflow of potassium ions. As shown schematically in Figure 2.3*B*, repetitive firing of neurons gives rise to increased extracellular potassium concen-

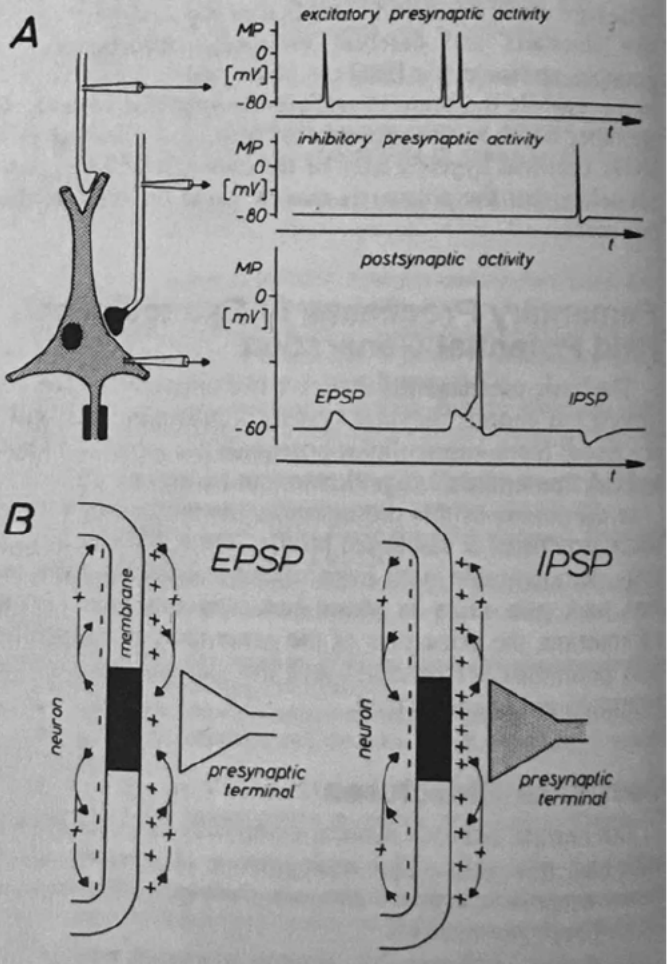

Figure 2.2. Membrane potential (MP) changes and current flows during synaptic activation. **A**, the MP of the postsynaptic neuron and the MP of the presynaptic fibers are recorded by means of intracellular microelectrodes. Action potentials in the excitatory and inhibitory presynaptic fiber lead to EPSP and IPSP, respectively, in the postsynaptic neuron. Two EPSPs sum up to a superthreshold potential, triggering an action potential in the postsynaptic neuron. **B**, during EPSP and IPSP, ionic current flows occur through as well as along the neuronal membrane, as shown by arrows. The density of + and - signs indicate the polarization of the subsynaptic (dark area) as well as that of the postsynaptic membrane during synaptic activation.

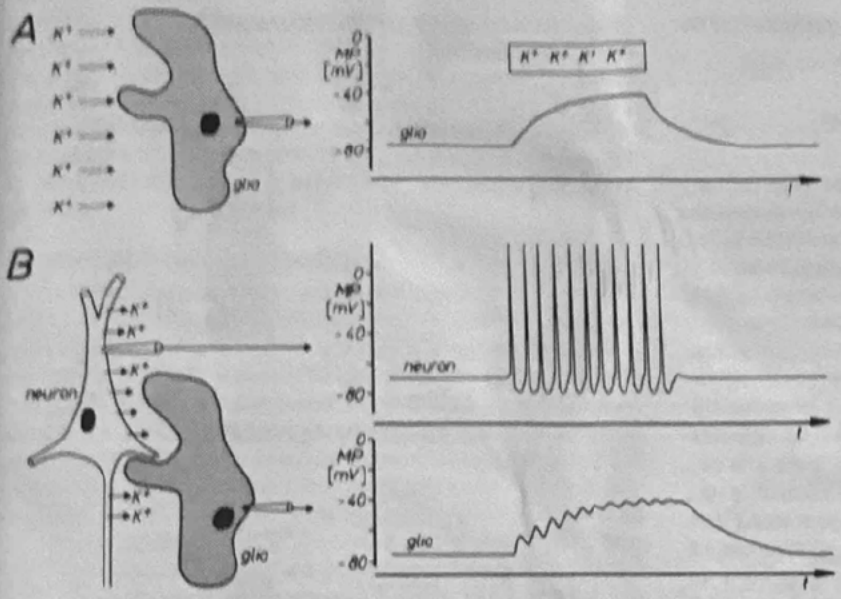

Figure 2.3. Membrane potential (MP) changes of glia cells induced by an increase in the extracellular K^+ concentration (arrows in the schematic drawings). **A**, potassium is applied extracellularly to the glia cell. **B**, the potassium concentration is increased due to an activation of a neighboring neuron. (Drawings after original tracings from Kuffler, S. W., Nicholls, J. G., and Orkand, R. K. 1966. Physiological properties of glial cells in the central nervous system of amphibia. J. Neurophysiol. 29:768–787.)

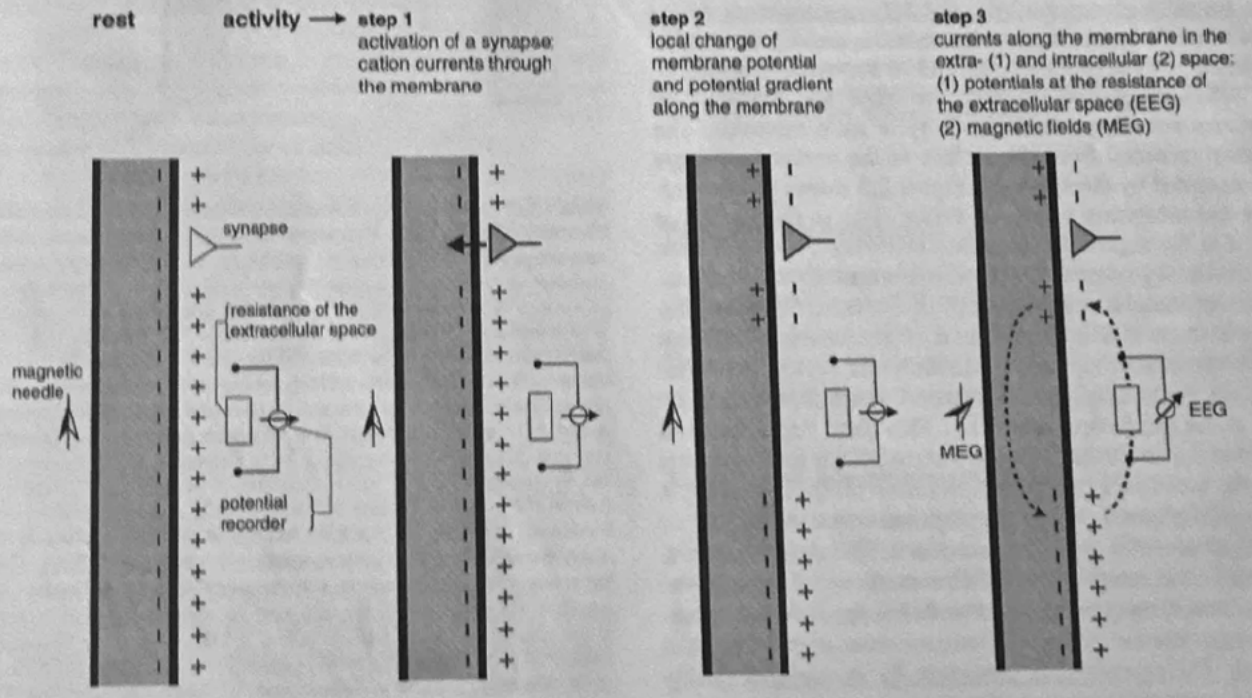

Figure 2.4. Basic mechanisms underlying generation of potentials (electroencephalogram; EEG) and of magnetic fields (magneto-encephalogram; MEG) in the extracellular space of central nervous system. The description is based on the assumption that an extended neuronal process, e.g., a dendrite, is locally depolarized by activation of an excitatory synapse.

tration and hence to glial cell depolarization (Orkand et al., 1966; Speckmann, 1986). If the potassium concentration does not affect the entire glial cell membrane and remains increased only locally, then potential gradients build up along the glial cell, giving rise to intra- and extracellular current flows similar to the ones described in reference to neuronal synaptic transmissions (Fig. 2.4). Glial cells frequently have widespread processes and furthermore may have close connections with each other. For this reason, potential fields of considerable spatial extension may develop on the basis of the aforementioned mechanisms (Caspers et al., 1980, 1984; Somjen and Trachtenberg, 1979; Speckmann and Caspers, 1979a). In view of the above described functional interconnections, it is quite likely that in the genesis of extracellula

field potentials an amplifying effect can be attributed to the glial cells.

Generation of Extracellular Field Potentials

It has been shown in the preceding section that primary transmembranous currents generate secondary ional currents along the cell membranes in the intra- and extracellular space. The portion of these currents that flows through the extracellular space is directly responsible for the generation of field potentials (Fig. 2.4). Particular significance must be ascribed to the synaptic processes as causing events for the field potentials in question, especially for their time course. In accordance with these statements, the generation of extracellular field potentials will be discussed as exemplified by extracellular fields accompanying synaptic activity (Caspers et al., 1984; Hubbard et al., 1969; Rall, 1977; Speckmann et al., 1984). The discussion of these events will again make use of a very protracted time axis (Fig. 2.4). The explanation of the events is given in reference to the schematic view in Figure 2.5. This figure shows a widely stretched neuronal element, with one end segment lying close to the surface of a central nervous structure. At both ends of this neuronal unit, the microelectrodes ME_1 and ME_2 are inserted. At the same time, the extracellular electrodes E_1 and E_2 are located at the surface and at the deeper end of the neuronal element. The potentials picked up from the intra- and extracellular electrodes are shown in the vicinity of each electrode. The potential recorded from the surface of the nervous structure is accentuated by *thicker lines*. Figure 2.5 shows active excitatory and inhibitory synapses, either close to the surface or located in the depth. As described elsewhere, the activation of an excitatory synapse leads to a net inward flow of cations. If this statement is applied to *A1* of Figure 2.5, then it becomes evident that the upper end of the neuronal element will be depolarized in comparison with other segments of the same cell. Accordingly, the synaptic current flow causes an EPSP at the microelectrode ME_1. This local depolarization then gives rise to further intra- and extracellular ionic currents along the nerve cell membrane. Because of the intracellular movements of positive charges, depolarization in the area of microelectrode ME_2 will also take place. This depolarization, however, is less steep and of smaller amplitude. At the superficially located extracellular electrode E_2, the inflow of positive charges into the neuronal element causes a negative field potential. The extracellular electrode E_2 is, metaphorically speaking, approached by positive charges so that a positive field potential will develop in this area. The point of reversal of the field potentials is localized between electrodes E_1 and E_2. The exact position of the point of reversal depends on the distribution of extracellular impedances.

Current flows of reversed direction (in reference to the recording electrodes) will occur if the active excitatory synapse is located at the deeper end of the neuronal element (Fig. 2.5, *A2*). In this case, positive charges approach the superficially located electrode (E_1) (again speaking metaphorically) and remove themselves from the deeply located electrode (E_2). This arrangement of the active synaptic structures causes a positive field potential at the surface and a negative one at the deep electrode. The current flows accompanying the activation of inhibitory synapses located in deeper and in more superficial areas, respectively, are shown in *B* of Figure 2.5. As can be derived from this illustration, the activation of a deep inhibitory synapse (Fig. 2.5, *B1*) produces a current flow that is largely similar to the one generated by the activation of a superficial excitatory synapse (Fig. 2.5, *A1*). In the same manner, there are also similar current flows in the extracellular space when a superficial

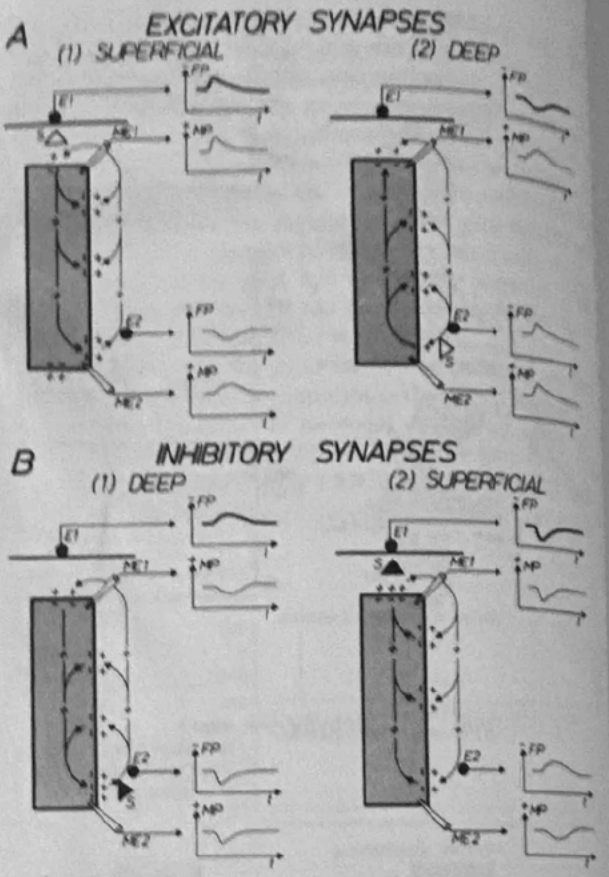

Figure 2.5. Membrane potential (MP) changes and field potentials (FP) elicited by the activation of excitatory and inhibitory synapses in the central nervous system. The elementary processes are explained by means of a neuronal element (hatched area), the one end of which contracts the surface of a structure in the central nervous system. The MP of the neuron element is recorded at both ends by the microelectrodes ME_1 and ME_2. The extracellular field is picked up at the surface of the neuronal structure by the electrode E_1, as well as in the vicinity of ME_2 by the electrode E_2. Active excitatory and inhibitory synapses are marked by open and black triangles (S), respectively. **A1**, the inward current at S generates an EPSP that appears in the region of ME_1, as well as in that of ME_2. Because S is located superficially, the FP generated is, due to the direction of the extracellular current flow (arrows), of negative polarity at the surface (E_1) and of positive polarity in the deeper recording (E_2). **A2**, the activation of a deep excitatory synapse elicits a current flow with inverse direction as compared with **A1**. Therefore, the extracellular FP consists in a positive deflection at the surface and in a negative one at the depth. **B1**, the outward current at S generates an IPSP in the region of ME_2, as well as in that of ME_1. Due to the direction of the extracellular current flow, the FP generated consists in a positive fluctuation in the depth (E2) and in a negative one in the surface recording (E1). **B2**, the current flow during the activation of a superficial inhibitory synapse is inverse as compared with **B1**. Therefore, the FP recorded from the surface consists of a positive fluctuation. Differences in the time course of the various potentials are caused by the electrical properties of the tissue.

inhibitory synapse (Fig. 2.5, *B2*) or a deeply located excitatory synapse (Fig. 2.5, *A2*) is activated. Accordingly, a negative field potential will develop at the surface of a central nervous structure (in the schematic view of Figure 2.5) whenever a superficial excitatory or a more deeply located inhibitory synapse is activated. The corresponding principle applies to generation of the superficial field potentials of positive polarity.

Types of Field Potentials

The field potentials, whose generation has been described, can be subdivided into different types. If field potentials are recorded against an inactive reference point with an upper frequency limit of about 100 Hz, then two types of field potentials can be distinguished, depending on the time constant of the amplifying recording device. In the case of a time constant of 1 second or less, the extracellular field potentials correspond with that which is commonly known as the electroencephalogram (EEG). If the recording is carried out with an infinite time constant, i.e., with a DC amplifier, then slower potentials can also be picked up. Potentials recorded with this technique are generally known as DC potentials (Caspers, 1974; Caspers et al., 1984; Speckmann and Caspers, 1979a; Speckmann et al., 1984). Thus, DC potentials comprise slow as well as fast field potentials. The fast components correspond with the potential fluctuations of the EEG. Due to different time constants, however, the faster potential components may differ from each other as far as their time course is concerned when recordings are done either with conventional EEG amplifiers or with DC amplifiers.

Thus far, technical problems have made it difficult to carry out DC recordings from the scalp. Except for special areas of application, DC recordings are usually performed in animal experiments. DC potentials directly reflect the state of activity of central nervous cells and therefore contribute to the explanation of the mechanisms of genesis of cerebral field potentials (Caspers et al., 1980; Speckmann and Caspers, 1979b). For this reason, DC potentials will be discussed jointly with EEG waves.

For the sake of comparison, Figure 2.6 shows the EEG and the DC potentials during convulsive activity, hypercapnia, and asphyxia. As shown in this illustration, a tonic-clonic convulsion is associated with a negative DC shift (Caspers and Speckmann, 1969; Caspers et al., 1980, 1984; Gumnit et al., 1970; Speckmann, 1986; Speckmann and Elger, 1984; Speckmann et al., 1984). Furthermore, it can be seen that the hypercapnia-induced disappearance of the EEG is associated with a monophasic positive DC shift. In the case of EEG extinction due to primary asphyxia, however, there are characteristic patterns of DC fluctuation. Hence, similar findings in the conventional EEG may be associated with different DC shifts.[2]

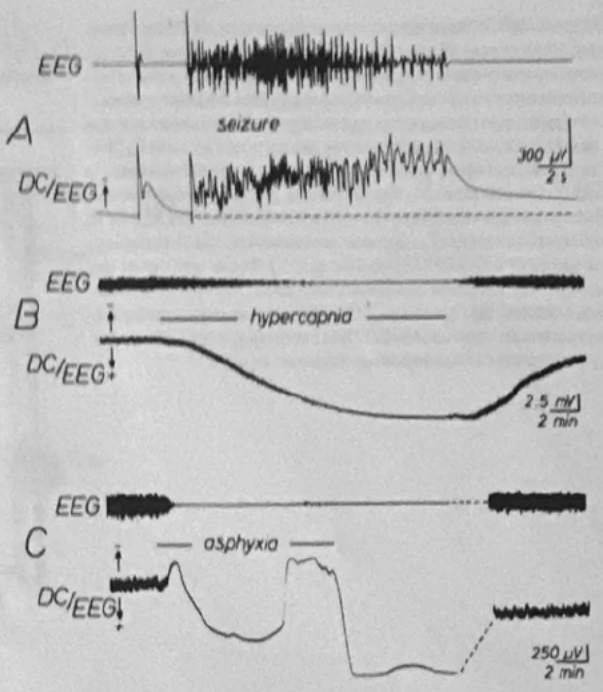

Figure 2.6. EEG (time constant: 1 second; upper frequency limit: 100 Hz) and DC/EEG recordings (DC recording: upper frequency limit—100 Hz) during a generalized seizure induced by pentylenetetrazol (**A**), during hypercapnia (**B**), and during asphyxia (**C**). Original recordings were obtained from cats and rats. Note the different time scales.

Wave Generation

In the preceding sections, the generation of single field potentials is described. In the following, the principles of the generation of wavelike potential fluctuations are outlined. This is followed by the discussion of the laminar distribution of such potentials in the cerebral cortex.

Principal Mechanisms

In order to present the generation of wavelike potential fluctuations on the surface of a central nervous structure, a simple model as shown in Figure 2.7 is used. This model consists of two extended pyramidal neurons of vertical orien-

[2] What does the term "DC shift" mean-what is "DC"? Speaking from experience, many electroencephalographers have no clear concept regarding DC potentials or DC shifts. One cannot blame them because, for strange reasons, "DC" has two meanings in this context.

1) (and this is, of course, commonplace): DC means direct current: a current without oscillations—a current derived from a battery source: a current maintained in one direction through a circuit. A more imperfect DC is produced by a rectifier (used to change alternating current [AC] into DC). For readers having problems with the English vocabulary: DC—direct current—is "courant continu" in French, "Gleichstrom" in German, "corrente continuo" in Italian.

As to the electroneurophysiological situation, DC shifts are ultraslow potentials—about as slow as 0,1–0.2/sec. This, however, is not true DC. Such slow activity is just a bit more "DC-like" since it does not show the faster "AC-like" activity. One simply has to live with this kind of misnomer.

2) (and this is much less known): DC also means "direct coupling." What coupling? The coupling between the stages of EEG amplification. Conventional EEG machines have stages coupled by capacitors. Now one has to remember that capacitors a) reject DC and b) determine the time constant. Even a very long time constant (several seconds duration) may not suffice for the recording of "DC potentials." "Direct coupling" is a capacitor-free coupling between the stages of amplification and provides the optimal condition for "DC recording." This is technically quite difficult in clinical conventional EEG recording but easier under experimental neurophysiological conditions in animals.

Hence, be aware of the dual significance of the term "DC" (also see Chapter 7 under "Filters"). (This footnote added by Ernst Niedermeyer, editor.)

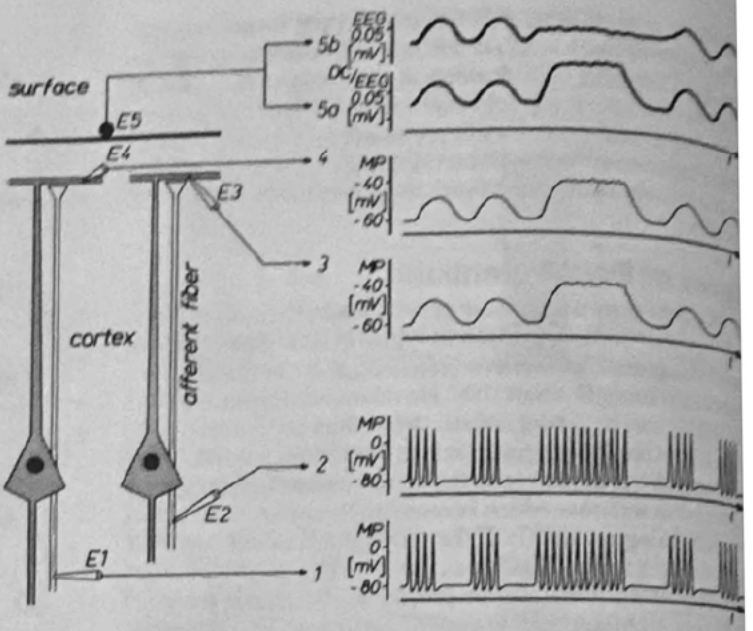

Figure 2.7. Principles of wave generation. The excitatory synapses of two afferent fibers contact the superficial dendritic arborization of two longitudinal neuronal elements. The afferent fiber activity is recorded by means of the intracellular electrodes E_1 and E_2, and the membrane potentials (MP) of the dendritic elements are recorded by the electrodes E_3 and E_4. The field potential at the surface of the neuronal structure (cortex) is led by the electrode E_5. Synchronized groups of action potentials in the afferent fibers (E_1, E_2) generate wavelike EPSPs in the dendritic areas (E_3, E_4) and corresponding field potentials in the EEG and DC/EEG recording (E_5). Tonic activity in the afferent fibers results in a longlasting EPSP with small fluctuations. During this period the EEG (5b) shows only a reduction in amplitude, whereas the DC/EEG recording (5a) reflects the depolarization of the neuronal elements as well.

tation. Terminals of afferent fibers make contact with the superficial dendrites of both neurons via excitatory synapses. The bioelectrical activity of these structures is recorded with intracellular microelectrodes. The microelectrodes E1 and E2 are located in the ascending fibers and the microelectrodes E3 and E4 are in the superficial dendrites of the postsynaptic neurons. In order to pick up the extracellular field potentials, the electrode E5 lies on the surface of the central nervous structure.

As shown in tracings 1 and 2, action potentials occur synchronously in the afferent fibers. There are grouped discharges that are temporarily supplanted by tonic activity. The ascending action potentials elicit individual EPSP in the upper dendrites of the neurons; these EPSPs are subsequently summated into major depolarizations in accordance with the discharge frequency. As shown in tracings 3 and 4, amplitude and duration of the depolarizations depend on the discharge pattern of the afferent fibers. The synaptic activity at the superficial structures gives rise to extracellular current flows resulting in superficial field potentials. With the use of DC recording techniques, the superficial field potentials reflect the potential fluctuations of the dendritic membrane. If, however, the superficial field potentials are recorded with a time constant of 1 second or less, then only the fast fluctuations of the superficial field potentials are demonstrable.

Thus far, the principles of genesis of EEG and DC waves have been shown in the schematic view of Figure 2.7. Accordingly, the generation of physiological EEG waves may be explained as follows. If a grouped and synchronous influx takes place in afferent fiber systems toward the superficial generator structures, then EEG waves evolve that are of high amplitude and distinctly separated from each other. In case of a periodic sequence of the afferent bursts, the recording of the field potentials shows sinusoidal potential fluctuations. This mechanism has been presumed by several groups of investigators as the principle of the generation of the alpha rhythm and slower periodic EEG waves. According to these workers, thalamocortical feedback loops are believed to play a significant role in the generation of the alpha rhythm (Andersen and Andersson, 1968; Speckmann and Caspers, 1979a).

If the afferent influx of impulses occurs at a high frequency for a longer period and/or synchronously, then negative field potentials with small fluctuations will result from the extracellular current flows. Accordingly, the EEG recording will pick up only waves of smaller amplitude and mostly higher frequency. In the DC recording, however, the prolonged depolarization of the superficial structures caused by the afferent high frequency influx will express itself by a negative DC potential shift (Caspers, 1963; Goldring, 1974). There is a close correlation between the amplitude of the negative DC shift and average discharge frequency in the afferent fiber systems. This mechanism may apply principally to the generation of beta activity and other EEG waves of higher frequencies. A decrease of the amplitudes of the EEG waves can also occur when the afferent activity is diminished. In this case, however, the depression of EEG waves is accompanied by a positive DC shift (Caspers and Speckmann, 1974, also see Fig. 2.14).

Spatial Distribution within the Cortex

The principles of generation of individual and wavelike field potentials at the surface of central nervous structures such as the cerebral cortex have been described. If the wavelike potential fluctuations are recorded not only from the cortical surface but also from different cortical layers, then it can be shown that potential fluctuations in the latter recordings may differ considerably from those at the surface. These differences imply polarity, frequency, and amplitude (Elger and Speckmann, 1983; Petsche et al., 1978; Speckmann and Caspers, 1979a). Such a recording from the cortex of the rat

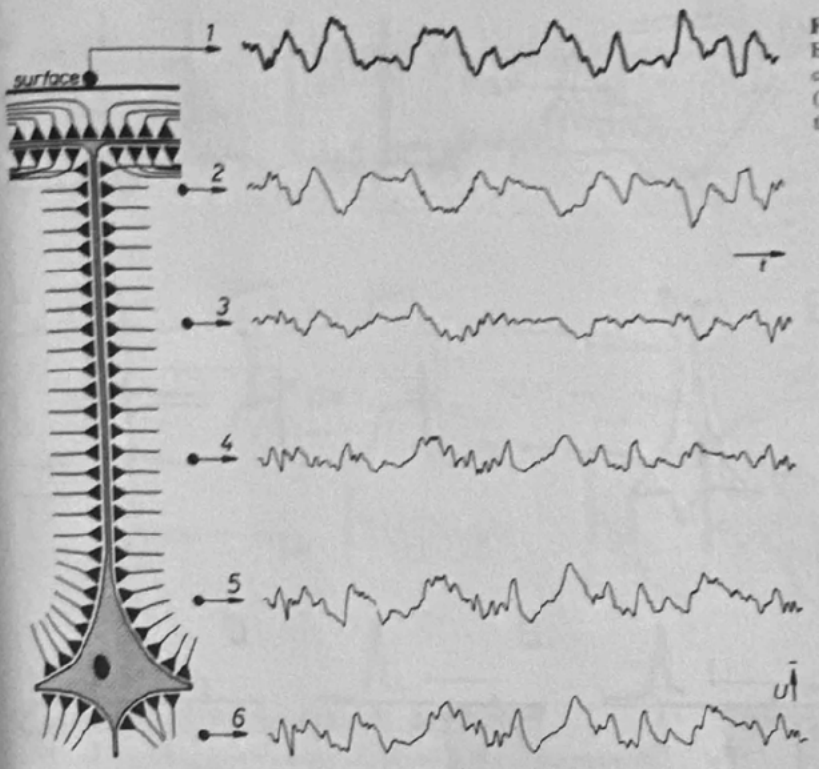

Figure 2.8. Surface (1) and laminar recordings (2–6) of EEG waves of the cortex. The schematic drawing symbolizes conical neuronal elements densely packed with synapses. (Drawings from original tracings obtained in experiments in the rat's motor cortex during pentobarbital anesthesia.)

is shown in Figure 2.8. According to this illustration, field potentials reverse their polarity between electrode 1 (on the surface) and electrode 2 (located 300 μm beneath the cortical surface). Two and sometimes more of such phase reversals may be observed in deeper recording sites depending on the experimental conditions. The vertical distribution type of field potential will be discussed in greater detail in connection with the generation of cortical field potentials during convulsive activity.

In the course of the discussion of cerebral field potentials, it was pointed out that particular significance must be attributed to synaptic activity. A view of the laminar distribution of neurons in the cortex and the dense coverage of these unitary structures with synapses makes it clear that different patterns of potentials must necessarily occur in different layers when populations of synapses are activated in a different manner. This should be clarified by the schematic drawing in Figure 2.8.

Cortical Field Potentials during Epileptiform Activity

In the following sections, the generation of cortical field potentials during convulsive activity is discussed. The first section deals with focal activity, and the second part discusses generalized, tonic-clonic convulsive activity. For methodical reasons, we will refer to data derived from experimental work in animals.

Focal Activity

If a convulsive substance such as penicillin is applied to the surface of the cerebral cortex, steep negative potentials of high amplitude can be picked up from the area of application after a short latency period. These discharges repeat themselves in stereotyped form and periodicity (Klee et al., 1982; Purpura et al., 1972; Speckmann, 1986; also see Fig. 2.9*A*). If the membrane potential of a cortical neuron is simultaneously recorded with a microelectrode while a second microelectrode picks up the corresponding field potentials, then potential fluctuations will occur as shown in Figure 2.9*B*. It can be derived from this illustration that the monotonously recurrent negative field potentials are associated with equally stereotyped membrane potential fluctuations. These oscillations of the membrane commence with a steep depolarization that, having exceeded the membrane threshold, will trigge a series of action potentials. This is followed by a platea that, after 80–100 msec, changes into a steep repolarizatio and frequently also into a hyperpolarization. These mem brane potential fluctuations have proved to be characteristi in the epileptiform activity of individual neurons. They a generally known as paroxysmal depolarization shifts (PDS (Jasper et al., 1969; Speckmann, 1986).

Investigation of potential distribution within the cerebr cortex after the local application of penicillin will yield variety of findings. An appropriate model is shown in Figu 2.10. In this experiment, recordings of interictal field pote tials were carried out from the cortical surface, from insi the cortex, and from the spinal cord. The spinal field pot tials permit the observation of electrical activity descendi from the cortex to the spinal cord. In Figure 2.10*A*, negat field potentials are recorded from the cortical surface from the two upper intracortical contacts after the applicat of penicillin together with penicillin-metabolizing enzy

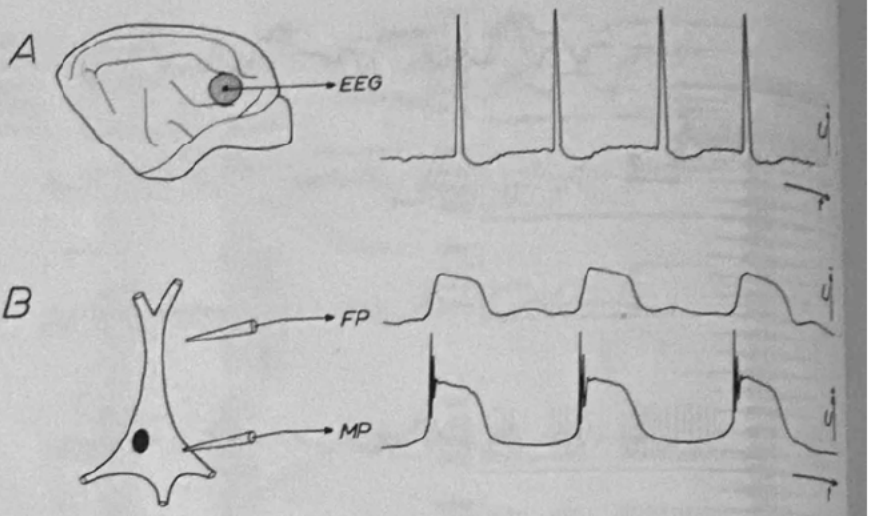

Figure 2.9. EEG (**A**) and membrane potential (MP) changes of a pyramidal tract neuron and extracellular field potential (FP) recorded in the vicinity of the impaled neuron (**B**) during focal interictal activity elicited by application of penicillin to the cortical surface (hatched area in **A**). Drawings of original tracings from experiments in the rat. The sweep speed in **B** is five times that in **A**. The recording sites are shown in the schematic drawings.

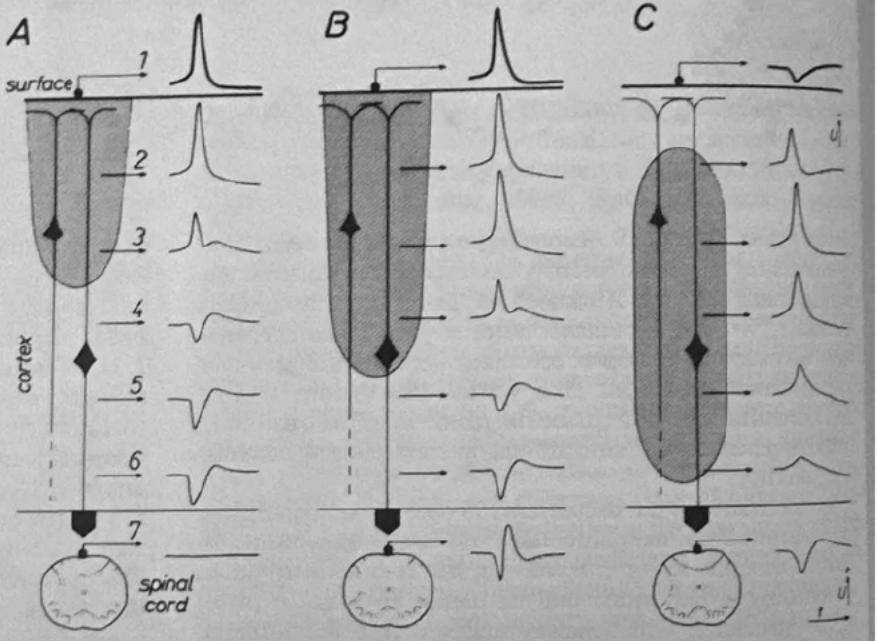

Figure 2.10. Cortical field potentials recorded at the surface (1) and from within the cortex (2–6) and spinal field potentials (7) during interictal activity. The interictal activity was elicited by penicillin. **A** and **B**, potential distribution after surface application of the drug. In **A**, the spread of penicillin is limited by the use of penicillinase. **C**, potential distribution after intracortical application of penicillin at recording point 4. The areas directly involved in the epileptiform activity as indicated by negative field potentials are marked by hatching in the schematic drawings. Spinal field potentials are linked to the occurrence of negative field potentials in lamina V (**B** and **C**, 4). Distance between the intracortical electrodes, 300 μm. (Drawings after original tracings from Elger, C.E., Speckmann, E.J., Caspers, H., and Prohaska, O. 1981. Focal interictal epileptiform discharges in the cortex of the rat: Laminar restriction and its consequences for activity descending to the spinal cord. In *Physiology and Pharmacology of Epileptogenic Phenomena*, Eds., M. R. Klee, H.D. Lux, and E.J. Speckmann. New York: Raven Press.)

penicillinase. There are, however, field potentials with predominantly positive components in the deeper contacts *4* through *6*. If penicillin is applied to the surface without penicillinase, then negative field potential will also develop in deeper cortical layers. If it is assumed that the negative field potentials mirror the direct epileptiform activity of neuronal structures (also see Fig. 2.9), then it must also be assumed that deeper cortical elements are involved in convulsive activity shown in *B* of Figure 2.10 in contrast with *A*. This is further supported by the observation that neuronal activity descending to the spinal cord and producing characteristic spinal field potentials occurs only under the experimental conditions shown in *B*. If one compares the recordings in *A* and *B*, it will become clear that, with a monotonous epileptiform potential at the cortical surface, the intracortical potential distribution and the occurrence of descending activity may differ considerably (Elger and Speckmann, 1980, 1983; Elger et al., 1981; also see Gumnit, 1974; Petsche et al., 1981; Speckmann and Elger, 1983; Wieser, 1983).

If penicillin is applied to deeper cortical laminae (Fig. 2.10*C*), then negative field potentials will be confined to that region. These potentials are consistently accompanied by descending activity to the spinal cord. Under these conditions, there is frequently nothing but a positive potential fluctuation of minor amplitude at the cortical surface (Elger and Speckmann, 1983; Elger et al., 1981).

In summary, it can be derived from the described experimental models that, in focal convulsive activity limited to the cortex, the surface potential does not necessarily reflect the bioelectrical events in deeper cortical layers.

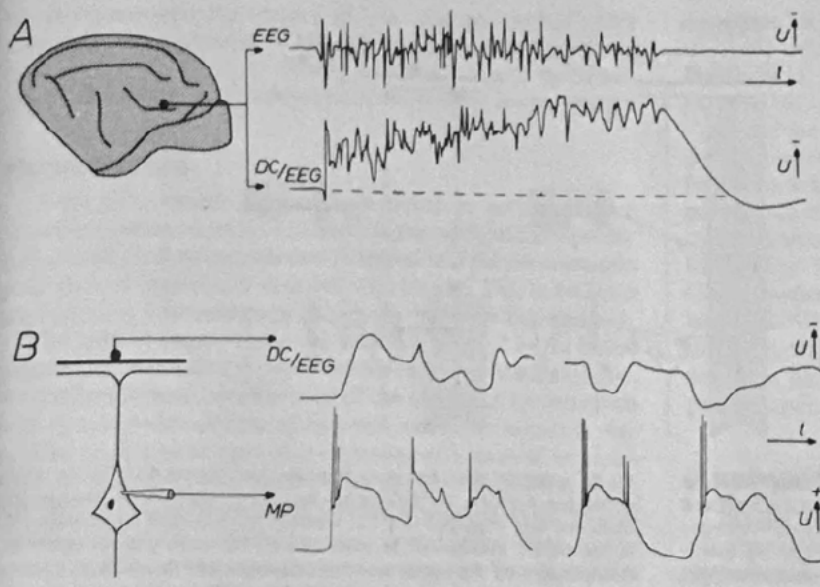

Figure 2.11. Simultaneous recordings of EEG and DC/EEG (**A**) and of DC/EEG and membrane potential (MP) of a pyramidal tract neuron (**B**) during generalized tonic-clonic seizures elicited by pentylenetetrazol. (Drawings after original tracings from experiments in the cat's motor cortex. The sweep speed in **B** is 10 times that in **A**.)

Generalized Tonic-Clonic Activity

Here, possible mechanisms involved in the generation of cortical field potentials during tonic-clonic convulsive activity are described. Again, data are based on experimental observations in the animal. Tonic-clonic convulsive activity was triggered by repeated injections of pentylenetetrazol (also see Purpura et al., 1972; Speckmann, 1986).

Figure 2.11*A* shows a tonic-clonic convulsion recorded with a conventional EEG amplifier, as well as with a DC amplifier. There is a negative DC shift from the baseline during a convulsive seizure. This negative DC shift gradually recedes during the termination of the convulsions and frequently changes into a transient positive after shift (Caspers and Speckmann, 1969; Caspers et al., 1980, 1984; Gumnit, 1974; Speckmann, 1986; Speckmann and Caspers, 1979b; Speckmann and Elger, 1984).

When the membrane potential of a pyramidal tract neuron of Lamina V is recorded during a convulsive seizure, it can be shown that under these conditions typical PDSs become manifest (Fig. 2.11*B*). If these PDSs are correlated with the potential fluctuations in the DC recording, it can be noticed that the PDS in pyramidal tract neurons are coupled at the beginning of the convulsive seizure with superficial negative potential fluctuations and at the end of the convulsive seizure with surface positive potential fluctuations (Figure 2.11*B*) (Speckmann et al., 1978; Speckmann and Caspers, 1979a, 1979b).

In addition to the field potentials of the cortical surface and the membrane potentials of the pyramidal tract cells, field potentials were also recorded in the fifth lamina. Under these conditions, it can be shown that every PDS is associated with a negative monophasic field potential in the depth (Fig. 2.12*A*). These stereotyped potential fluctuations in deep cortical layers correspond with field potentials at the cortical surface with either monophasic negative or positive (Fig. 2.12, *A1* and *3*) or with polyphasic (Fig. 2.12, *A2*) configurations.

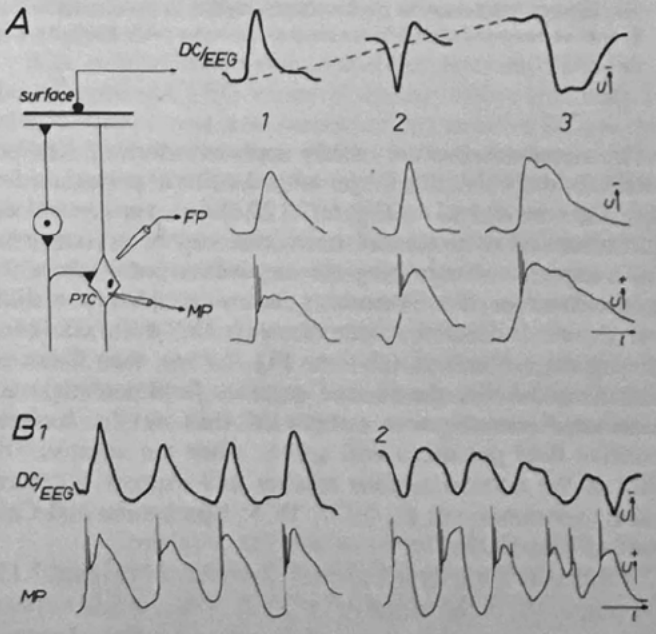

Figure 2.12. Single potential fluctuations at the cortical surface (DC/EEG and concomitant membrane potential (MP) of a pyramidal tract cell (PTC and field potentials (FP) in the PTC layer during generalized tonic-cloni seizures. The seizure activity was induced by pentylenetetrazol. **A**, the nega tive potential (1), the positive-negative fluctuation (2), and the positive p tential (3) in the DC/EEG recording coincide with monophasic negative F and stereotyped paroxysmal depolarization shift in the neuron. The negati DC shift occurring during the seizure is indicated by a dashed line in t upper row. Monophasic negative potentials in the DC/EEG recording occ with small and monophasic positive fluctuations along with a marked I displacement. **B**, the relations between DC/EEG potentials and MP of P as described for A1 and A3 also hold true for trains of potentials (1, (Drawings after original tracings from Speckmann, E.J., Caspers, H., Jansen, R.W.C. 1978. Laminar distribution of cortical field potentials relation to neuronal field activities during seizure discharges. In *Architect ics of the Cerebral Cortex*, IBRO Monograph Series, vol. 3, Eds., M. Brazier and H. Petsche, pp. 191–209. New York: Raven Press.)

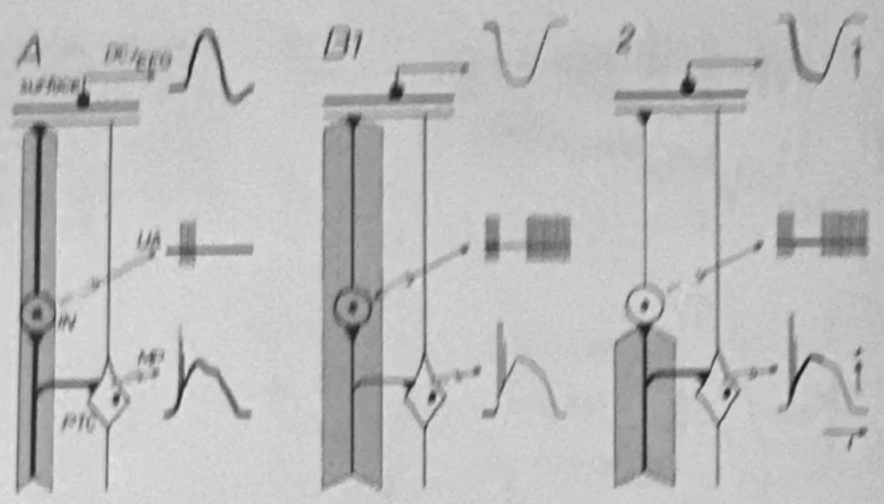

Figure 2.13. Flow charts of neuronal processes possibly responsible for the generation of DC/EEG waves of opposite polarity during a generalized tonic-clonic seizure. Hatched arrows, symbols for continuous asynchronous input to the cortex; heavy lines, symbols for phasic volleys giving rise to single convulsive discharges; PTC, pyramidal tract cell; IN, interneuron; MP, membrane potential; UA, extracellularly recorded unit activity. **A**, during a moderate asynchronous input to the cortex (small hatched arrow), a burst of UA triggers a paroxysmal depolarization shift in a PTC. Simultaneously, it leads to a depolarization of superficial neuronal structures and therewith to a negative fluctuation in the DC/EEG recording at the cortical surface. **B**, with an increased asynchronous input to the cortex (wide hatched arrow), the DC potential shifts to a more negative level than in **A** (1). When in these conditions a phasic volley reaches the cortex, paroxysmal depolarization shifts are also triggered in PTC, whereas the enhanced asynchronous UA is interrupted mainly due to inactivation. The latter process results in a disfacilitation of the upper neuronal structures and therewith to a positive fluctuation of the superficial DC/EEG potential (2). (Drawings of original tracings from Speckmann, E.J., Caspers, H., and Jansen, R.W.C. 1978. Laminar distribution of cortical fluid potentials in relation to neuronal field activities during seizure discharges. In *Architectonics of the Cerebral Cortex*, IBRO Monograph Series, vol. 3, Eds., M.A.B. Brazier and H. Petsche, pp. 191–209. New York: Raven Press.)

This statement does not merely apply to individual ictal potentials but is also true for prolonged trains of potentials during the convulsion. As Figure 2.12*B* shows, paroxysmal depolarizations of pyramidal tract cells may be accompanied by a sequence of either negative or positive potentials on the cortical surface. If one correlates these various field potentials on the cortical surface with the slow DC shifts occurring during the convulsion (also see Fig. 2.12*A*), then it can be demonstrated that the surface-negative field potentials are associated primarily with a slight DC shift and that surface-positive field potentials will appear when the negative DC shift at the cortical surface reaches and exceeds a critical value (Speckmann et al., 1972, 1978; Speckmann and Caspers, 1979a, 1979b).

These data are interpreted with flow charts in Figure 2.13. The amplitude of the negative DC shift at the cortical surface depends greatly on the amount of the afferent influx of impulses to the generator structures in the superficial cortical laminae. This predominantly asynchronous afferent influx is symbolized by the width of hatched arrows in Figure 2.13. Accordingly, the afferent influx in *A* of Figure 2.13 is smaller than that in *B*. Therefore, there is a smaller DC shift in *A* and a prominent one in *B*. In the case of *A*, a synchronized inflow of impulses from subcortical structures is assumed to reach the cortex (widened afferent fiber in schematic view). As a consequence, pyramidal tract cells will be stimulated to generate a PDS and structures close to the surface will be depolarized through the mediation of interneurons. Accordingly, in such a constellation of excitatory processes, the paroxysmal depolarization in the depth will be coupled with a surface-negative field potential. With augmentation of the already existing afferent inflow of impulses, the interneurons involved will necessarily exhibit a heightened level of excitation (Fig. 2.13*B*). If an additional highly synchronized afferent influx of impulses takes place under these conditions, then further PDS will be triggered in the pyramidal tract cells, but, in the interneurons, the previously existing high frequency activity will be temporarily interrupted, chiefly due to inactivation. This will cause a decline of the excitatory inflow of impulses to the superficial cortical structures. This disfacilitation gives rise to a positive field potential at the cortical surface. In this manner, a massive afferent inflow of impulses provides the basis for a correlation of positive epicortical field potentials with stereotyped paroxysmal depolarizations and monophasic negative field potentials in the depth (Speckmann et al., 1978; Speckmann and Caspers, 1979b).

Cortical Field Potentials during Gas Tension Changes in Tissue

The following sections deal with the alterations of epicortical field potentials and concomitant changes of the membrane potentials caused by deviations of the gas tension in brain tissue. Such changes of the gas tension may occur when, for instance, the pulmonary and circulatory function is disturbed or when the local cerebral blood flow is inadequate.

First, the alterations of epicortical field potentials during selective hypercapnia are discussed; then, those associated with primary asphyxia are considered. It is shown that EEG changes may be similar under both conditions. The cortical DC potential, however, shows typical shifts that permit infer-

ences concerning the cause of the accompanying EEG changes. The discussion of the effects of gas tension alterations on the bioelectrical activity of the CNS will be based, again, on data derived from experimental work in the animal.

Hypercapnia

If the CO_2 tension in the brain tissue is increased in a selective manner, typical reactions of the cortical field potentials as well as of the membrane potential and the postsynaptic potentials of individual neurons are found. These findings are shown in a summarized schematic view in Figure 2.14.

The animal experiments on which Figure 2.14 is based were carried out with the use of the so-called apnea technique. With this technique, interference of the effects of hypercapnia with simultaneous effects of hypoxia could be avoided. According to this technique, the experimental animal is ventilated for at least ½ hour with pure oxygen. Thereafter, artificial ventilation is discontinued while the trachea of the animal remains connected with the O_2 reservoir. Under these conditions, the CO_2 tension progressively rises in the tissue for about 15 minutes without a concomitant fall of the oxygen tension below the baseline level.

With isolated increment of the CO_2 tension in the cerebral tissue by means of the apnea technique, the amplitude of the conventional EEG decreases progressively. This amplitude reduction affects first the waves of higher frequency and then those of lower frequency. Prior to the extinction of normal EEG activity, there is once again a phase characterized by high frequency EEG activity in the range of 50–70 Hz (Caspers et al., 1979; Speckmann and Caspers, 1979a). The extinction of the EEG is associated with a shift of the DC potential in a positive direction. If the CO_2 tension is then lowered again by reventilation, the EEG waves return in the original spectral composition after a short latency. At the same time, the positive DC shift resolves (Fig. 2.14). Experiments in animals have shown that, with reduction of the pCO_2, the EEG returns to normal activity even though the hypercapnia-induced suppression lasted for 1 hour or more. In these cases, a positive DC deflection of monophasic character was found to occur during the whole period of apnea (Caspers and Speckmann, 1974; Caspers et al., 1979; Speckmann and Caspers, 1974).

Under the aforementioned conditions, the recording of the membrane potential of a cortical nerve cell shows a hyperpolarization while the CO_2 tension is increased. Extensive experimental studies in animals have demonstrated that such a hyperpolarization is caused primarily by a reduction of the EPSP (Fig. 2.14; also see Speckmann and Caspers, 1974). Consideration of field potentials, of membrane potentials, and of EPSP shows that epicortical DC potentials reflect neuronal hyperpolarization. The disappearance of the EEG waves is presumed to be caused mainly by the reduction of postsynaptic activity.

Asphyxia

Primary asphyxia exemplified by respiratory arrest after air ventilation is associated with combined CNS effects of hypercapnia and hypoxia. The effects of gas tension changes on the field potentials and on the membrane potential of individual neurons are schematically shown in Figure 2.15. In the corresponding animal experiments, the artificial ventilation with air was either temporarily (*A*) or persistently (*B* interrupted.

With such an interruption of artificial ventilation with air the conventional EEG waves disappear within less than minute. This process is accompanied by a negative DC potential shift from the baseline, which has been characterized as ''initial negativity'' (see under *1* in Fig. 2.15). While the EEG shows an isoelectric line in the further course of asphyxia additional potential shifts are detectable with DC recording technique. The initial negativity is followed by a positive DC shift termed ''intermediate positivity'' (see under 2 in Fig. 2.15). If reventilation is performed in this phase of asphyxia an additional positive DC shift is observed, appropriately termed ''reactive positivity'' (see under *3* in Fig. 2.15). According to the analysis of the experimental work, the int

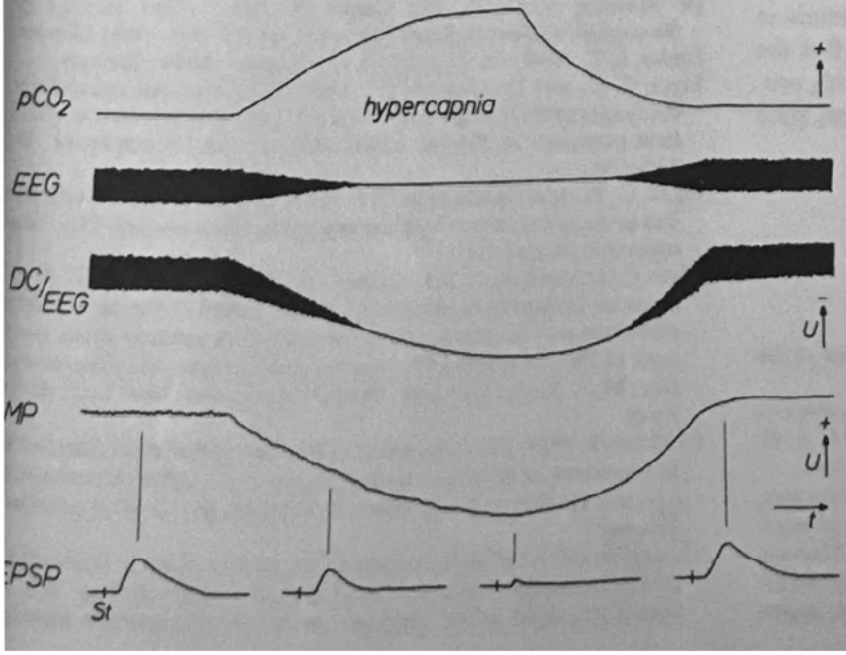

Figure 2.14. Effects of an isolated hypercapnia on epicort field potentials (EEG, DC/EEG) and on membrane pote (MP). With increasing pCO_2, the EEG disappears even if pO_2 is above normal levels. The disappearance of the EE associated with a positive DC shift and a hyperpolarizatic most of the neurons. Simultaneously, the amplitudes of stim (St) evoked EPSP are markedly reduced. (Drawings after c nal tracings from Speckmann, E.J., and Caspers, H. 1974. effect of O_2 and CO_2 tensions in the nervous tissue on neu activity and DC potentials. In *Handbook of Electroenceph raphy and Clinical Neurophysiology*, vol. 2, Part C, Ed.-in- A. Remond, pp. 71–89. Amsterdam: Elsevier.)

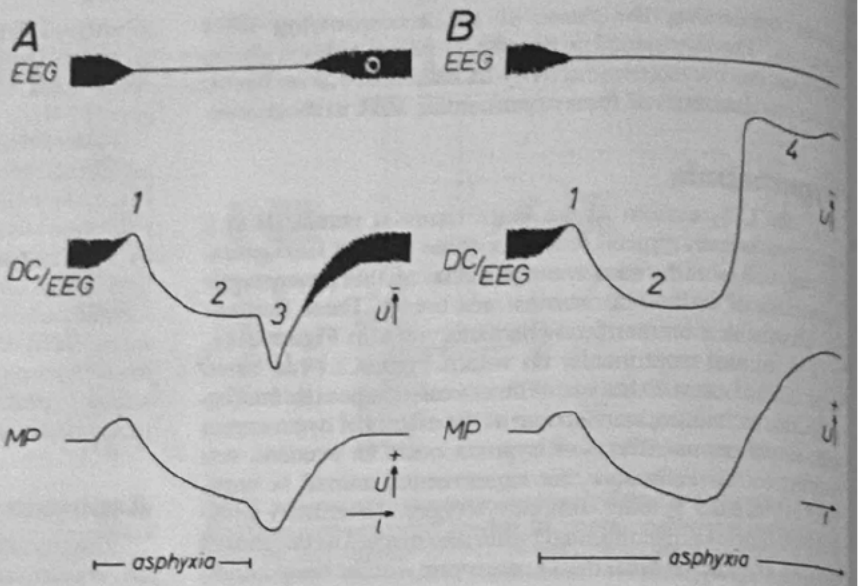

Figure 2.15. Alterations of EEG, DC/EEG, and neuronal membrane potential (MP) during primary asphyxia. **A**, the abolition and the reappearance of EEG during a transient asphyxia goes in parallel with typical DC shifts: (1) initial negativity, (2) intermediate positivity, (3) reactive positivity. These DC fluctuations are accompanied by corresponding reactions of the MP. **B**, with continuing asphyxia, the EEG remains abolished and the intermediate positivity (2) turns over into a terminal negativity (4). The latter DC negativity corresponds to a breakdown of neuronal membrane potential. (Drawings after original tracings from Speckmann, E.J., and Caspers, H. 1974. The effect of O_2 and CO_2 tensions in the nervous tissue on neuronal activity and DC potentials. In *Handbook of Electroencephalography and Clinical Neurophysiology*, vol. 2, Part C, Ed.-in-chief, A. Remond, pp. 71–89. Amsterdam: Elsevier.)

mediate and the reactive types of positivity are due to an increase of CO_2 tension in the brain tissue. With the resolution of the reactive positivity, a restitution of the fast field potentials occurs that is also demonstrable with the conventional EEG. A comparison of the DC shifts and the alterations of the membrane potentials shows a parallelism of both events (Caspers and Speckmann, 1974; Caspers et al., 1979, 1980, 1984; Speckmann and Caspers, 1974, 1979a).

If the interruption of the artificial ventilation is continued for a longer period of time, then the intermediate positivity converts into the so-called "terminal negativity" (see under *4* in Fig. 2.15*B*). This negative DC shift correlates with the breakdown of the neuronal membrane potential. The terminal effects are due to a critical lack of oxygen. The terminal negativity may be reversible for a substantial period of time under certain experimental conditions if the artificial ventilation is resumed and the reduction of the cerebral circulation is counteracted with circulation support measures (Speckmann and Caspers, 1974).

In summary, a comparison of EEG and DC potentials in selective hypercapnia and primary asphyxia shows that the recording of cortical field potentials with DC amplifiers provides a more accurate picture of the actual functional state of nerve cells.

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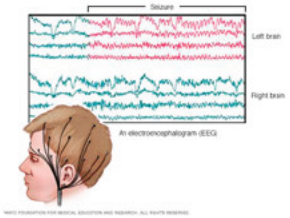
EXHIBIT 3

EEG (electroencephalogram)

Overview



EEG brain activity

Enlarge image

An electroencephalogram (EEG) is a test that measures electrical activity in the brain using small, metal discs (electrodes) attached to the scalp. Brain cells communicate via electrical impulses and are active all the time, even during asleep. This activity shows up as wavy lines on an EEG recording.

An EEG is one of the main diagnostic tests for epilepsy. An EEG can also play a role in diagnosing other brain disorders.

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Why it's done

An EEG can find changes in brain activity that might be useful in diagnosing brain disorders, especially epilepsy or another seizure disorder. An EEG might also be helpful for diagnosing or treating:

- Brain tumors
- Brain damage from head injury
- Brain dysfunction that can have a variety of causes (encephalopathy)
- Sleep disorders
- Inflammation of the brain (herpes encephalitis)
- Stroke
- Sleep disorders
- Creutzfeldt-Jakob disease

An EEG might also be used to confirm brain death in someone in a persistent coma. A continuous EEG is used to help find the right level of anesthesia for someone in a medically induced coma.

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Absence seizure

Amnesia

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Risks

EEGs are safe and painless. Sometimes seizures are intentionally triggered in people with epilepsy during the test, but appropriate medical care is provided if needed.

How you prepare

Food and medications

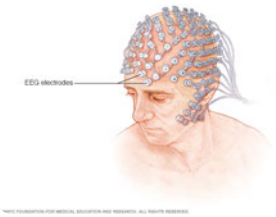
Take your usual medications unless instructed otherwise.

Other precautions

- Wash your hair the night before or the day of the test, but don't use conditioners, hair creams, sprays or styling gels. Hair products can make it harder for the sticky patches that hold the electrodes to adhere to your scalp.
- If you're supposed to sleep during your EEG, your health care provider might ask you to sleep less or avoid sleep the night before your EEG.

What you can expect

During the test



EEG electrodes
Enlarge image

You'll feel little or no discomfort during an EEG. The electrodes don't transmit any sensations. They just record your brain waves.

Here are some things you can expect to happen during an EEG:

- **A technician measures your head and marks your scalp** with a special pencil to indicate where to attach the electrodes. These spots on your scalp might be scrubbed with a gritty cream to improve the quality of the recording.
- **A technician attaches discs (electrodes) to your scalp** using a special adhesive. Sometimes, an elastic cap fitted with electrodes is used instead. The electrodes are connected with wires to an instrument that amplifies the brain waves and records them on computer equipment.

  Once the electrodes are in place, an EEG typically takes between 20 and 40 minutes. Testing for certain conditions requires you to sleep during the test. In that case, the test can be longer.

- **You relax in a comfortable position with your eyes closed during the test.** At various times, the technician might ask you to open and close your eyes, perform a few simple calculations, read a paragraph, look at a picture, breathe deeply for a few minutes, or look at a flashing light.
- **Video is routinely recorded during the EEG.** Your body motions are captured by a video camera while the EEG records your brain waves. This combined recording can help your doctor diagnose and treat your condition.

Ambulatory EEGs (aEEGs) allow for longer monitoring outside an office or a hospital setting. But these types of EEGs aren't always an option. This test can record brain activity over several days, which increases the chances of recording during seizure activity. However, compared with inpatient video EEG monitoring, an ambulatory EEG is not as good at determining the difference between epileptic seizures and nonepileptic seizures.

After the test

The technician removes the electrodes or cap. If you didn't have a sedative, you should feel no side effects after the procedure. You should be able to return to your typical routine.

If you used a sedative, it will take time for the medication to begin to wear off. Arrange to have someone drive you home. Once you're at home, rest and don't drive for the rest of the day.

Results

Doctors trained to analyze EEGs interpret the recording and send the results to the doctor who ordered the EEG. You might need to schedule an office appointment to discuss the results of the test.

If possible, bring along a family member or friend to the appointment to help you remember the information you're given.

Write down questions to ask your doctor, such as:

- Based on the results, what are my next steps?
- What follow-up, if any, do I need?
- Are there factors that might have affected the results of this test in some way?
- Will I need to repeat the test?

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